

A palpable mass is unusual in patients with tuberculous peritonitis when there is no accompanying gastrointestinal involvement. This finding has been noted in 0% to 4% of cases in several American series,^{3,5-7} though two reports from third world nations describe a 30% incidence of palpable masses.^{4,8} Perhaps this reflects the later stage at which tuberculous peritonitis first comes to medical attention in countries where health care resources are scarce.

The diagnosis of tuberculous peritonitis is problematic when it presents as an abdominal mass. Many patients have no clinical evidence of tuberculosis elsewhere, and peritoneoscopy is hazardous because of the extensive adhesions.⁹ Needle aspiration or biopsy under CT guidance may yield the diagnosis, but laparotomy is sometimes necessary, as in the case described above.

Epstein and Mann recently described the CT findings in eight South African patients with intra-abdominal tuberculous masses.¹⁰ Four patients had irregular soft-tissue densities in the omentum, three had low-density masses surrounded by thick, solid rims and two had low-density lymph nodes with rim enhancement. These hypodense areas probably represent foci of caseous necrosis.¹⁰ Dahlene and co-workers reported the cases of three patients with tuberculous peritonitis in whom the CT scan showed irregular thickening and nodularity of the mesentery, but no masses.¹¹ The radiographic findings in our case are similar to those described above, and the combination of an abdominal mass with a low-density center and mesenteric adenopathy should suggest the possibility of mycobacterial infection. It should be kept in mind that this CT appearance may be mimicked by two other processes: one is bacterial mesenteric adenitis with abscess formation, and the second is lymphoma with extensive central necrosis as a result of chemotherapy or radiation treatment. Percutaneous aspiration of an abdominal mass is usually possible under CT guidance, and appropriate stains of the aspirated material for mycobacteria should always be done so that laparotomy may be avoided.

Mycobacterial infection is a common complication of AIDS. The high prevalence of atypical mycobacterial disease has been frequently emphasized in the literature, though in our experience, tuberculosis is more common than atypical mycobacteriosis in these patients (Peter Barnes, MD, unpublished data, June 1984 to April 1985). This is probably a result of the high prevalence of tuberculosis in the population served by our hospital.

When tuberculosis occurs in patients with AIDS, extrapulmonary involvement is seen in 70% of cases.¹ Though our patient had subclinical pulmonary tuberculosis manifest only by positive sputum cultures, the most clinically significant problem was peritoneal infection. This predilection for extrapulmonary disease is probably due to widespread hematogenous dissemination of mycobacteria in patients with deficient cell-mediated immunity. Despite their immunodeficiency, patients with AIDS respond very well to antituberculous drugs. It is a crucial diagnosis to make, therefore, because many other infections and tumors in these patients are not as readily treatable.

There is preliminary evidence to suggest that tuberculosis often precedes the development of Kaposi's sarcoma or opportunistic infections, whereas atypical mycobacteria cause disease after the full-blown manifestations of immunodeficiency have developed.¹ Presumably, this is due to the fact that *M tuberculosis* is intrinsically more virulent than the atypical forms and therefore results in infection at an earlier stage of immunodeficiency. It is interesting that our patient has had no opportunistic infections to date, and his case may be an example of this phenomenon. The large number of acid-fast bacilli seen in his peritoneal tissue is unusual in tuberculosis and probably reflects his defective cell-mediated immunity.

REFERENCES

1. Pitchenik AE, Cole C, Russell BW, et al: Tuberculosis, atypical mycobacteriosis and the acquired immunodeficiency syndrome among Haitian and non-Haitian patients in south Florida. *Ann Intern Med* 1984; 101:641-645
2. Barrow DW: Tuberculous peritonitis. *South Med J* 1943; 36:646-650
3. Kahrs T: Tuberculous peritonitis—A follow-up study of 619 cases. *Tubercle* 1952; 33:132-138
4. Borhanmenesh F, Hekmat K, Vaezzadeh K, et al: Tuberculous peritonitis—Prospective study of 32 cases in Iran. *Ann Intern Med* 1972; 76:567-572
5. Burack WR, Hollister RM: Tuberculous peritonitis. *Am J Med* 1960; 28:510-523
6. Karney WW, O'Donoghue JM, Ostrow JH, et al: The spectrum of tuberculous peritonitis. *Chest* 1977; 72:310-315
7. Sochocky S: Tuberculous peritonitis—A review of 100 cases. *Am Rev Respir Dis* 1967; 95:398-401
8. Novis BH, Banks S, Marks IN: Gastrointestinal and peritoneal tuberculosis. *S Afr Med J* 1973; 43:365-372
9. Singh MM, Bhargava AN, Jain KP: Tuberculous peritonitis—An evaluation of pathogenetic mechanisms, diagnostic procedures and therapeutic measures. *N Engl J Med* 1969; 281:1091-1094
10. Epstein BM, Mann JH: CT of abdominal tuberculosis. *AJR* 1982; 139:861-866
11. Dahlene DH, Stanley RJ, Koehler RE, et al: Abdominal tuberculosis: CT findings. *J Comput Assist Tomogr* 1984; 8:443-445

The Acquired Immunodeficiency Syndrome and Hypercalcemia

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THE acquired immunodeficiency syndrome (AIDS) is characterized by severe immunologic dysfunction (cell-mediated more than humoral) and manifested by a multitude of opportunistic infections, unusual neoplasms and autoimmune phenomena.¹ Using a combination of an enzyme-linked immunosorbent assay (ELISA) and the electrophoretic (Western blot) technique, antibodies to a type C retrovirus (usually human T-cell leukemia/lymphoma virus III, or HTLV-III) can be shown in almost all such cases.^{2,3} Although hypercalcemia is a common complication in patients with a T-cell lymphoproliferative syndrome associated with a different type C retrovirus (HTLV-I),⁴ this metabolic abnormality has only recently been reported to occur in patients with AIDS.⁵ An additional case showing this association is herein presented and the possible underlying pathophysiology discussed.

Report of a Case

The patient, a 31-year-old sexually active homosexual man, had a five-month illness characterized by an 18-kg (40-lb) weight loss, low-grade fevers, night sweats and pro-

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ABBREVIATIONS USED IN TEXT

AIDS = acquired immunodeficiency syndrome
 CMV = cytomegalovirus
 ELISA = enzyme-linked immunosorbent assay
 HTLV-I, -III = human T-cell leukemia/lymphoma virus I or III

gressive weakness. His condition was initially evaluated in August 1984 by his local physician. Relevant laboratory studies showed a leukocyte count of 3,200 per μ l with 4% lymphocytes. Tests were positive for antibody to hepatitis B core and surface antigens. A serum protein electrophoresis showed a polyclonal hypergammaglobulinemia. The serum calcium level was 10.3 mg per dl and repeated was 8.9 mg per dl. Serum albumin and phosphorus levels were normal. Skin tests to *Candida*, histoplasmosis, coccidioidomycosis and purified-protein-derivative antigens were nonreactive. T-cell subsets measured in September 1984 showed total T cells of 274 (normal 1,200 to 1,800), OKT4-positive (helper-induced) cells 67 (normal 600 to 1,200), OKT8-positive (suppressor) cells 207 (normal 350 to 600) and a helper-to-suppressor ratio of 0.32 (normal > 0.85).

On October 2, 1984, the patient presented to the Stanford Hospital emergency department in acute respiratory distress. He was taking no medications. His temperature taken orally was 40°C (104°F) and the respiratory rate was 40 breaths per minute. His fundi were normal. He had white plaques typical of candidiasis on his oral mucosa. His lungs were clear. He had no lymphadenopathy, hepatosplenomegaly or skin lesions. Admission laboratory studies elicited the following values: leukocyte count 3,200 per μ l with 73% segmental forms, 14% bands, 7% metamyelocytes and 4% lymphocytes, 2% eosinophils; serum calcium 12.5 mg per dl, phosphorus 4.3 mg per dl, creatinine 1.1 mg per dl, albumin 3.1 grams per dl and alkaline phosphatase 79 IU per liter. A chest roentgenogram showed a bibasilar reticulonodular infiltrate with relative sparing of the upper lobes. The patient underwent immediate bronchoscopy and bronchoalveolar lavage. The washings were smear-positive for *Pneumocystis carinii*, and cultures grew *Candida albicans*.

A regimen of trimethoprim, 240 mg, and sulfamethoxazole, 1,200 mg, given intravenously every six hours, was begun. Serum calcium levels remained elevated between 11.8 mg per dl and 13.6 mg per dl, despite saline and furosemide diuresis and administering calcitonin, 200 IU, intramuscularly every 12 hours. Corticosteroid therapy was not tried for fear of worsening the patient's already severely compromised immunologic state. On the eighth hospital day, 0.75 mg of plicamycin (formerly mithramycin) was given intravenously, and by the tenth hospital day the serum calcium level began to fall but never returned to normal. Concomitant values for serum phosphorus and albumin continued normal until the last five days of life, when the patient had hypoalbuminemia (1.5 to 1.8 grams per dl). The serum free thyroxine level was normal at 1.4 ng per dl. An 8 AM plasma cortisol level was normal at 22 μ g per dl. A serum parathormone level (N-terminal; American Bio-Science) was less than 150 pg per ml (normal 230 to 630), with a serum ionized calcium of 5.4 mEq per liter (normal 4.4 to 5.2). A serum vitamin D level was not measured. Tests for antibodies to both HTLV-I and HTLV-III retroviruses (by the ELISA method) were negative.

The hospital course was complicated by a progressive deterioration in the patient's respiratory state despite intubation and the addition on the eighth hospital day of 200 mg per day of pentamidine isethionate given intramuscularly. The patient died on the 13th hospital day.

A postmortem examination showed severe *P. carinii* pneumonia and lymphocyte depletion and plasmacytosis of lymph nodes and spleen. There was no evidence of cytomegalovirus (CMV), granulomatous disease, parathyroid abnormalities or neoplasms of any kind.

Discussion

Although antibodies to HTLV-III retrovirus can almost always be shown in patients with AIDS, the diagnosis of this condition remains a clinical one.⁶ Our patient satisfied these clinical criteria and had the typical pathologic findings as well.⁷

The cause of this patient's hypercalcemia is unknown. There was no pathologic or biochemical evidence for adrenal or thyroid abnormalities. He received no medication(s) to account for this finding nor was there evidence for malignancy at postmortem examination. Zaloga and co-workers suggested that their patients with AIDS had elevated serum calcium levels from increased osteoclastic bone resorption resulting from cytomegalovirus infection.⁵ Unlike their cases, however, our patient did not have infection with CMV, nor did the hypercalcemia respond to calcitonin therapy. Clues to other possible mechanisms may be extrapolated from studies of certain other hypercalcemic states. For example, it has been postulated that the elevated serum calcium level associated with adult T-cell lymphoma is due to neoplastic T-cell production of a lymphokine resembling osteoclast-activating factor.⁴ It has also been shown that certain patients with adult T-cell lymphoma and hypercalcemia have elevated calcitriol levels.^{8,9} A similar finding has been shown to be responsible for the hypercalcemia seen in patients with various granulomatous diseases.¹⁰ The patients of Zaloga and associates, however, had low serum calcitriol levels. We did not measure this hormone in our patient.

It is likely that despite the negative antibody reaction to HTLV-III, our patient was nevertheless infected with this virus, since antibody titers (as determined by the ELISA method) may decline during the clinical course of AIDS.³ HTLV-III virus is characterized by substantial genetic polymorphism and probably represents a set of viruses with closely related but varying genetic forms.² Rather than implicating a secondary infectious agent as the cause of an elevated serum calcium level in patients with AIDS (CMV, as Zaloga and colleagues postulate), it is more likely that the pathogenic retrovirus might express itself as its relative in the HTLV-I group and cause hypercalcemia by an as yet unknown mechanism.

Although this case only raises questions and answers none, it does serve to expand the clinical presentation of AIDS and should alert clinicians to the potentially serious associated metabolic abnormality of hypercalcemia.

REFERENCES

1. Fauci AS, Macher AM, Longo DL, et al: Acquired immunodeficiency syndrome: Epidemiologic, clinical, immunologic and therapeutic considerations. *Ann Intern Med* 1984; 100:92-106
2. Broder S, Gallo RC: A pathogenic retrovirus (HTLV-III) linked to AIDS. *N Engl J Med* 1984; 311:1292-1297

3. Weiss SH, Goedert JJ, Sarngadharan MG, et al: Screening test for HTLV-III (AIDS agent) antibodies. *JAMA* 1985; 253:221-249
4. Broder S, Bunn PA, Jaffe ES, et al: T-cell lymphoproliferative syndrome associated with human T-cell leukemia/lymphoma virus. *Ann Intern Med* 1984; 100:543-557
5. Zaloga GP, Chernow B, Eil C: Hypercalcemia and disseminated cytomegalovirus infection in the acquired immunodeficiency syndrome. *Ann Intern Med* 1985; 102:331-333
6. Council on Scientific Affairs: The acquired immunodeficiency syndrome: Commentary. *JAMA* 1984; 252:2037-2043
7. Welch K, Finkbeiner W, Alpers CE, et al: Autopsy findings in the acquired immune deficiency syndrome. *JAMA* 1984; 252:1152-1159
8. Breslau NA, McGuire JL, Zerwekh JE, et al: Hypercalcemia associated with increased serum calcitriol levels in three patients with lymphoma. *Ann Intern Med* 1984; 100:1-7
9. Rosenthal N, Insogna KL, Goddard JW, et al: Elevations of circulating 1,25-dihydroxyvitamin D in three patients with lymphoma-associated hypercalcemia. *J Clin Endocrinol Metab* 1985; 60:29-33
10. Lemann J, Gray RW: Calcitriol, calcium and granulomatous disease (Editorial). *N Engl J Med* 1984; 311:1115-1117

Bronchiolitis Obliterans Due to *Mycoplasma pneumoniae*

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BECAUSE the definitive diagnosis of bronchiolitis obliterans requires lung biopsy, the occurrence of this disorder in various diseases has not been well described. Considered rare in adults, bronchiolitis obliterans has been associated with toxic gas inhalation, connective tissue disorders and postinfectious states.^{1,2} In many cases a cause cannot be determined, and it is often presumed to be due to viral infection.³⁻⁶

We report this case because *Mycoplasma pneumoniae* has been implicated as a cause of bronchiolitis obliterans⁷ but only rarely confirmed pathologically.^{8,9} We present the case of a man in whom respiratory failure developed and he was found to have bronchiolitis obliterans. Cold-agglutinin titers were elevated and he improved on a regimen of erythromycin. Subsequent complement-fixation titers supported the diagnosis of *M pneumoniae* infection.

Report of a Case

The patient, a 37-year-old man, was transferred to the University of New Mexico Hospital on May 16, 1983, with a

three-week history of fever, earache, nonproductive cough and progressive shortness of breath.

He stopped smoking in February 1983 at the request of his children. He had worked as a welder but had been unemployed for several months. He said he had no recent exposure to toxic gas or fumes. His health was good until about May 1, when he had a fever to 39.4°C (103°F), and an earache and a nonproductive cough developed. At the same time, his 13-year-old son had a similar illness. For the next week the patient treated himself with penicillin without consulting a physician. When shortness of breath developed, however, he went to a local emergency room and cefaclor therapy was started. On May 11 he was admitted to a local hospital because of worsening dyspnea. At that time he had a leukocyte count of 25,000 per μ l, with 80% segmented cells, 12% bands, 2% lymphocytes and 6% monocytes. The hematocrit was 46%. Analysis of arterial blood gases with the patient breathing room air at an altitude of 1,810 m showed the following values: pH 7.50, partial pressure of arterial oxygen (Pao₂) 28 torr and partial pressure of arterial carbon dioxide (Paco₂) 30 torr. A chest roentgenogram showed minimal bibasilar nodular infiltrates (Figure 1). Empiric treatment was begun with cefotaxime sodium given intravenously. Cultures of sputum and blood specimens taken on admission were negative for pathogens. After four days his condition was not improving and arterial blood gas determinations with the patient breathing room air showed a pH of 7.48, a Pao₂ of 26 torr and a Paco₂ of 42 torr. An empiric trial of prednisone, 80 mg a day, was started. Five days after hospital admission, arterial blood gas determinations with the patient breathing 40% oxygen by mask showed a pH of 7.45, Pao₂ 42 torr and Paco₂ 44 torr. Because of his severe hypoxemia, he was transferred to the University of New Mexico Hospital.

On admission to the University of New Mexico Hospital, the patient's blood pressure was 148/96 mm of mercury, pulse 80 beats per minute, respiratory rate 22 per minute and temperature 37.4°C (99.3°F) orally. His breathing was labored. The tympanic membranes were normal. There were crackles in both lung bases without wheezes. An S₃ gallop was present with normal first and second sounds. The abdomen was not tender and the liver and spleen were not enlarged. There was no pedal edema.

With the patient receiving oxygen via a nonrebreathing mask, analysis of arterial blood gases showed a pH of 7.43, a Pao₂ of 53 torr and a Paco₂ of 62 torr. His chest roentgenogram was unchanged. Spirometry the following day (May 17) showed a severe obstructive ventilatory defect (Table 1). He was unable to produce sputum. A cold-agglutinin titer was 1:512. A regimen of erythromycin given intravenously was

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TABLE 1.—Course of Pulmonary Function Tests

	5-17-83	5-26-83	6-27-83	9-26-83	Predicted
Forced vital capacity, liters	1.44	1.95	3.0	3.89	4.76
FEV ₁ , liters	0.96	1.43	2.2	2.70	3.95
MMEF 50, liters/s	...	1.05	...	2.95	5.77
Residual volume, liters	...	2.56	...	2.82	1.68
Total lung capacity, liters	...	4.51	...	6.53	6.38
D _{lco} , ml/min/mm of mercury	38.3	36.3

FEV₁ = forced expired volume in one second, MMEF 50 = maximal midexpiratory flow at 50% of forced vital capacity, D_{lco} = diffusing capacity for carbon monoxide